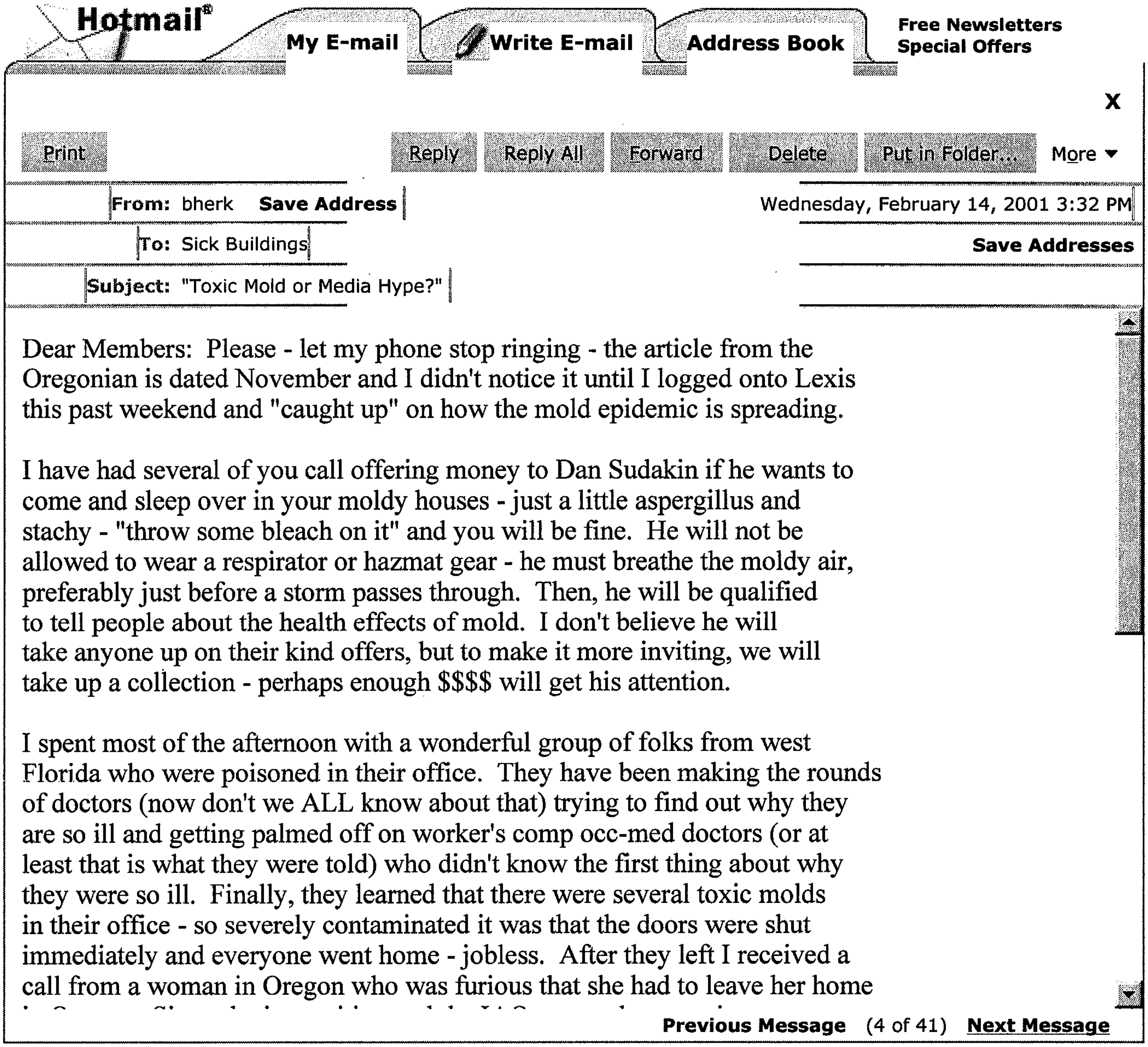

From: bherk

To: Sick Buildings

Subject: "Toxic Mold or Media Hype?"

Wednesday, February 14, 2001 3:32 PM

Dear Members: Please - let my phone stop ringing - the article from the Oregonian is dated November and I didn't notice it until I logged onto Lexis this past weekend and "caught up" on how the mold epidemic is spreading.

I have had several of you call offering money to Dan Sudakin if he wants to come and sleep over in your moldy houses - just a little aspergillus and stachy - "throw some bleach on it" and you will be fine. He will not be allowed to wear a respirator or hazmat gear - he must breathe the moldy air, preferably just before a storm passes through. Then, he will be qualified to tell people about the health effects of mold. I don't believe he will take anyone up on their kind offers, but to make it more inviting, we will take up a collection - perhaps enough $$$$ will get his attention.

I spent most of the afternoon with a wonderful group of folks from west Florida who were poisoned in their office. They have been making the rounds of doctors (now don't we ALL know about that) trying to find out why they are so ill and getting palmed off on worker's comp occ-med doctors (or at least that is what they were told) who didn't know the first thing about why they were so ill. Finally, they learned that there were several toxic molds in their office - so severely contaminated it was that the doors were shut immediately and everyone went home - jobless. After they left I received a call from a woman in Oregon who was furious that she had to leave her home

(4 of 41)

343.747/affidavit of daniel lee sudakin

NO.99-05252

MARY MELINDA BALLARD and § **IN THE DISTRICT COURT**
RONALD ALLISON, individually and §
As Next Friends for Reese Allison, A Minor, §
§
Plaintiffs, §
§
VS. § **345TH JUDICIAL DISTRICT**
§
FIRE INSURANCE EXCHANGE, §
A MEMBER OF THE FARMERS §
INSURANCE GROUP AND §
FRANK HRUSKA D/B/A §
HRUSKA INSURANCE AGENCY, §
§
Defendants, § **TRAVIS COUNTY, TEXAS**

AFFIDAVIT OF DANIEL LEE SUDAKIN, M.D., M.P.H.

STATE OF OREGON§
§
COUNTY OF BENTON §

BEFORE ME, the undersigned authority, on this day personally appeared DANIEL LEE SUDAKIN, M.D.; M.P.H., who being duly sworn, did depose and say:

1. "My name is Daniel Lee Sudakin, M.D., M.P.H. I am of sound mind, and am fully qualified and capable of making this Affidavit. I am over the age of 21 and have never been convicted of a crime of moral turpitude.

2. "I have been licensed to practice medicine in the state of Oregon since June, 1996. I have been certified by the American Board of Preventive Medicine (Public Health & General Preventive Medicine) since January, 2000. I obtained my Doctor of Medicine in May, 1994 from Wayne State University School of Medicine in Detroit, Michigan. In 1995, I completed my internal medicine internship at Legacy Hospital in Portland, Oregon. I completed a clinical anesthesiology internship in 1996 at Stanford University Health Services in Palo Alto, California. In 1998, I completed my residency in Public Health & General Preventive Medicine (Occupational & Environmental Medicine Track) at Oregon Health Sciences University in Portland, Oregon. In June, 1999, I completed the Master of

Public Health Program at Oregon Health Science University in Portland, Oregon. From 1998 to 2000, I completed a Medical Toxicology Fellowship at the Veterans Administration Medical Center in Portland, Oregon. I am board certified in the subspecialty of medical toxicology by the American Board of Preventive Medicine.

3. "My professional experience includes my service as an expert panel member on the Centers for Disease Control & Prevention Environmental/Laboratory Investigation panel for Acute Idiopathic Pulmonary Hemorrhage in August of 2000. This position involved my substantial review of the literature on toxigenic molds and other environmental risk factors that could possibly contribute to pulmonary hemorrhage in infants. I was an adjunct instructor at the Occupational Health Clinics of Oregon Health Sciences University from April 1999 to June 2000. I have been a member of the Drug Utilization Review Council in the Oregon Medical Assistance Programs from 1997 to December, 2000. I was also an abstractor for the Centers for Disease Control Guide to Community Preventive Services from 1997 to 1998. I am currently an assistant professor in the Department of Environmental and Molecular Toxicology at Oregon State University.

4. "My publications include:

a. Abstract presentations, North American Congress of Clinical Toxicology, September, 2000, entitled
"Regional Variation in the Incidence of Intentional Drug & Chemical Exposures"
"Plumbism and Pregnancy Treated with DMSA" and
"Lead Chelation in the Neonate;"

b. Special Article, "Stachybotrys chartarum: Current Knowledge of its Role in Disease," Medscape General Medicine Journal, February 29, 2000;

c. Case Report, "Intermediate Syndrome After Malathion Ingestion Despite Continuous Infusion of Pralidoxime." Journal of Toxicology, Clinical Toxicology 2000; 38(1):47-50;

d. Letter to the Editor, "A National Survey of Regional Poison Control Centers' Management of Occupational Exposure Calls." Journal of Occupational and Environmental Medicine, 2000; 42(1):1-2;

e. Letter to the Editor, "Mycotoxins and Pulmonary Hemorrhage." Archives of Pediatrics and Adolescent Medicine 1999; 153:205;

f. Abstract Presentations, North American Congress of Clinical Toxicology, September, 1999, entitled
"Geographic Information Systems and Poison Center Management"
"Geographic Distribution of Hospitals and their influence on Poison Center Penetrance" and

"Intermediate Syndrome After Malathion Ingestion Despite Continuous Infusion of 2-PAM";

g. Letter of the Editor, "Integrating Preventive Medicine in a Medical Resident Practice" Archives of Internal Medicine, August 10/24, 1998;

h. Original Manuscript, "Toxigenic Fungi in a Water-Damaged Building: An Intervention Study." American Journal of Industrial Medicine 1998; 34:183-90;

i. Letter to the Editor, "Effects of Mycotoxins in Health and Disease." Journal of the American Medical Association. October 1, 1997.

5. "I am a member of the American College of Medical Toxicology and the American College of Occupational & Environmental Medicine. I am also a member of the Northwest Association of Occupational & Environmental Medicine, The American Medical Association, and Alpha Omega Alpha, a medical honorary fraternity.

6. "I have participated as a consulting and treating physician in the evaluation and management of hundreds of patients with hazardous occupational and environmental exposures. Many of these patients have reported exposure to toxigenic mold. As a physician practicing within the subspecialty of medical toxicology, I am experienced in collecting a detailed medical history, performing a complete physical examination, and analyzing laboratory results obtained from individuals with toxic exposures.

7. "In forming my opinions expressed herein, I have reviewed the following documents:

I. Medical records from:
   a. Wayne Gordon, Ph.D. (Ron and Reese Allison)
   b. Eckhart Johanning, M.D. (Ron and Reese Allison)
   c. Dr. Drew Sawyer (Reese Allison)
   d. Austin Open MRI (Ron Allison)
   e. Dr. Peter Werner (Ron Allison)
   f. Dr. Laurence Ligon (Ron Allison)
   g. Dr. Bennie McWilliams (Reese Allison)
   h. Dr. Sarmistha Hauger (Reese Allison)
   i. Dr. Michael Bayer (Reese Allison)
   j. P&K Microbiology Services (Ron Allison)
   k. IBT Reference Laboratories (Ron Allison)
   l. Specialty Laboratories (Ron and Reese Allison)
   m. Clinical Pathology Labs (Ron and Reese Allison)
   n. Dr. David Pampe (Ron Allison)
   o. Dr. Mark Clark (Ron Allison)
   p. Dr. Bradley Fullerton
   q. Brackenridge Hospital (Reese Allison)

II. Records from:
  a. Assured Indoor Air Quality
  b. Microbiology Specialists
  c. Jan Koehn, M.S., CIH
  d. Texas Tech University Health Science Center
  e. Rimkus Consulting Group – Dr. Dan Bridge
  f. Armstrong Laboratories
  g. Aerotech Laboratories

III. Deposition transcripts of:
  a. Melinda Ballard
  b. Ron Allison
  c. Dr. J. Van Bavel (and medical records attached as exhibits)
  d. Dr. David Straus
  e. Homer (Bill) Holder
  f. J.D. Cooley
  g. Dr. Wayne Gordon
  h. Daniel Bridge, Ph.D.
  i. Dr. Eckhart Johanning

IV. Appraisal documents from Plaintiffs and Defendants;

V. Plaintiff's Third Amended Petition; and

VI. Plaintiffs' Supplemental Document Production of November, 2000.

8. a. "Eckardt Johanning's medical evaluation of Ron Allison is unreliable. It does not provide convincing evidence of exposure to mycotoxins, and does not include evaluation for several other potential etiologies for Mr. Allison's health concerns. There are important inconsistencies in Mr. Allison's medical screening questionnaire (completed by Mr. Allison) and Dr. Johanning's medical record. In the questionnaire, Mr. Allison acknowledges that he is an occasional cigar smoker. In the medical record, Dr. Johanning reports that Mr. Allison has never smoked. An accurate smoking history is of critical importance in the clinical evaluation of any patient with pulmonary complaints. In his patient questionnaire, Mr. Allison also reported drinking alcohol occasionally. Dr. Johanning does not include this in the medical report. The documentation of an accurate alcohol history is of obvious importance in the clinical evaluation of a patient with complaints of cognitive problems and "blackouts" as described by Mr. Allison in his questionnaire. Mr. Allison's medical screening questionnaire reveals that there is a pet bird in the household. Exposure to pet birds is known to be a potential risk factor for the development of severe pneumonia and other health problems in humans. This is another environmental risk factor not included in Dr. Johanning's differential diagnosis. In addition to these inconsistencies between Mr. Allison's reported history and what is documented in the medical record, Dr. Johanning did

not conduct a neurological examination of Mr. Allison. The medical record contains Dr. Johanning's assessment of Mr. Allison's mental status, but no report of his strength, sensation, reflexes, or cranial nerve function. These tests are commonly performed as part of a standard physical examination, and are of critical importance to a physician evaluating a patient for a toxic exposure. A diagnosis of encephalopathy, from any etiology, cannot be valid without a complete physical examination of the neurological system.

b. "With respect to the epidemiological studies upon which Dr. Johanning relies for the propostion that exposure to toxic molds causes lung disease and brain damage in the general population, these studies have fundamental flaws in their design and methodology. In fact, the case-control study relied upon by Dr. Johanning has been criticized in several peer-review scientific journals, including the New England Journal of Medicine, because of concerns that selection and reporting bias may have influenced the data. In a properly designed case-control study, cases are defined by a medical diagnosis, and controls by definition do not have that diagnosis. The investigator then compares the cases and controls for the amount of exposure they have had to a hypothesized risk factor. In the case-control study relied upon by Dr. Johanning, cases were defined by their duration of employment in a building, without respect to whether a case had any symptoms. All cases were defined as having some exposure to mold (although the data reflect that some had no exposure to Stachybotrys mold), while all controls were assumed to have no exposure (yet no effort was made to validate this assumption). This assumption with respect to controls is a false one, as it is known that mold is a ubiquitous exposure in the indoor and outdoor environment, and that molds like *Stachybotrys*, *Aspergillus*, and *Penicillium* are found in the homes of healthy individuals, especially when using "aggressive" sampling techniques as was the method in this case-control study. The definition of exposure is further complicated by the fact that the physical presence of a mold in an environment does not necessarily indicate that an exposure to mycotoxins has taken place. Thus, in this case-control study, there was significant misclassification bias between cases and controls, a systematic mislabeling of cases as exposed and controls as unexposed. In reality, it is entirely unclear whether cases or controls received any dose of mycotoxins, or what the differences in dose may have been between the two groups. When considering that several of the "case" subjects were Dr. Johanning's patients, and pursued litigation against the owner of the building under investigation (a limitation not discussed in the original publication), there is clearly a potential for recall bias to have influenced the reporting of symptoms among "cases". Only one objective finding (a blood test of questionable clinical relevance) was found to have a statistically significant difference (P-value less than .05) between cases and controls in this study. This was one of over 20 comparisons of different blood tests that were made between cases and controls. This finding could easily be attributable to making multiple comparisons, and for this reason, the appropriate epidemiological approach to interpreting this finding would be to adjust the P-value downward (far less than .01) to ensure the result were not a chance observation. The authors made no adjustment for multiple comparisons in this case-control study.

9. a. "The laboratory tests upon which Dr. Johanning relies are not sufficient to establish human exposure to mycotoxins. After a review of the test reports and correspondence, I have determined that two different tests, also known as "assays," were utilized.

b. "The first of these tests is known as the MTT cleavage test, in which the kidney cells of pigs are exposed to an environmental sample or some purified form thereof. In this particular instance, the samples from the Ballard house were tested against one control, which exposed filter paper to the pig kidney assay. Both the Ballard specimens and the control (filter paper) tested positive for toxicity, making the results inconclusive. This test provides no information on whether a sample contains mycotoxins. It does not distinguish between the effects of other co-existing contaminants (such as dust, fiberglass, paint residue, or heavy metals such as lead) in a sample that could cause toxicity to the kidney cells of pigs. Using this testing method, one might conclude that filter paper is a toxic substance to pig kidney cells, although the identity or chemical structure of the substance responsible for the toxicity cannot be determined. The use of this particular test is described in a paper co-authored by Dr. Johanning (Mycotoxin Cytotoxicity Screening of Field Samples, pg. 202), in which 44 homes with mold contamination whose occupants were complaining of health problems were tested. In 21 of these cases, no cytotoxicity was detected using the MTT cleavage test, despite the fact that molds like Stachybotrys, Aspergillus, and Penicillium were encountered in 38%, 57%, and 76% of samples, respectively. If the hypothesis is that these health complaints were related to mycotoxin exposure, these findings raise obvious doubts regarding the extent to which this test has any meaningful correlation with toxicity in humans. This test is not accepted within the scientific community or approved by the Food and Drug Administration as a valid or reliable method to measure dose or confirm human exposure to mycotoxins.

c. "The second test utilized on the Ballard samples was a luciferase protein translation assay. This test involved exposing rabbit reticulyte cells to the environmental samples. A toxic reaction would inhibit the function of luciferase, which is the protein that causes a lightning bug's tail to glow. Toxicity is indirectly measured by how much light inhibition is observed. Similar to the MTT bioassay, this test does not confirm whether mycotoxins are present in a sample, thus any number of different environmental contaminants could be responsible. The experimental application of the luciferase assay was the subject of a recent scientific publication, in which the toxicity of samples of Stachybotrys collected from the homes of sick infants were compared to samples of Stachybotrys that were present in the homes of healthy infants. Using the luciferase assay, the investigators found that the strains of Stachybotrys with the highest amount of measured toxicity came from the homes of healthy infants. This clearly raises important questions regarding the extent to which this test correlates with human disease. The luciferase assay is not accepted by the scientific community, nor approved by the Food and Drug Administration, as a valid or reliable method to confirm human exposure to mycotoxins.

d. "In using this test to conclude that the Plaintiffs suffered mycotoxin exposure, the experts relied upon a false assumption when they stated in their report that, "Although no straight forward process has been developed to pull mycotoxins from the air, it is safe to correlate at this point that if filter extracts from a house contaminated with *Stachybotrys* species demonstrate the ability to inhibit protein synthesis, then mycotoxins are in the house air system." There is no scientific basis for this assumption, as the luciferase assay does not provide any information on the chemical structure or identity of the substance responsible for the toxicity.

10. "There are scientific methods that can reliably detect the presence of mycotoxins in mold or other environmental samples. None of these methods have been utilized in the evaluation of the Plaintiffs' complaint. Even when mycotoxins are detected in a sample, this does not mean that human exposure has taken place. In the scientific literature, it has been shown using reliable methods of detection (high performance liquid chromatography) that some environmental samples with the highest quantity of mycotoxins have been obtained from the homes of healthy infants.

11. "Without a reliable biological marker to indicate the presence of mycotoxin within the human body, it is impossible to quantify the dose of mycotoxin to which a person may be exposed, or predict the response to that dose.

12. "There is disagreement within the scientific community as to whether the relatively large size of Stachybotrys spores prevents it from penetrating to the deepest areas of the lung. If the mold spore cannot reach this area of the lung, then the biological plausibility of toxigenic molds as a cause of lung injury (pulmonary hemorrhage) from inhalation exposure is limited. In addition, the mycotoxins produced by Stachybotrys (known as trichothecenes) have a high molecular weight, and for this reason would not be expected to volatilize (become airborne) independent of the mold spores. Indeed, trichothecenes have never been detected in indoor air using reliable methods of detection (high performance liquid chromatography). This is an additional factor that limits the biological plausibility of inhalation effects from exposure to trichothecene mycotoxins.

13. "Further, affiant sayeth naught."

Daniel Lee Sudakin MD, MPH
DANIEL LEE SUDAKIN, M.D., M.P.H.

SWORN AND SUBSCRIBED to me under oath on this the 22 day of January 2001, 200~~0~~1 LS

OFFICIAL SEAL
LINDA LEATHAM
NOTARY PUBLIC-OREGON
COMMISSION NO. 301709
MY COMMISSION EXPIRES JUN 02, 2001

Linda Leatham
NOTARY PUBLIC, State of Oregon
County of Benton

James,

Thanks Tear it apart please!

8 pgs.

EXHIBIT "A"

D. SUDAKIN